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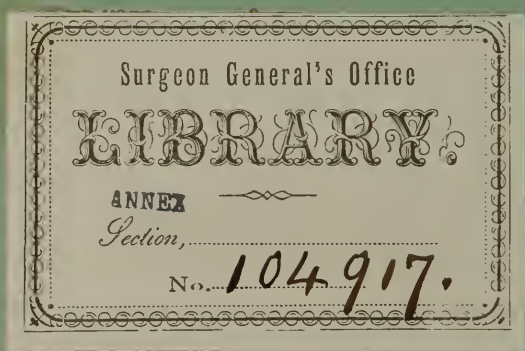
PHYSICAL DIAGNOSIS
— OF —
THORACIC DISEASES

E. DARWIN HUDSON, JR.



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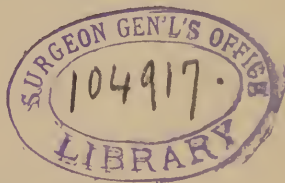


ESSENTIALS
OF
THE PHYSICAL DIAGNOSIS
OF
THORACIC DISEASES.

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Prepared for the use of the Physicians in the Class of General Medicine and
Diseases of the Chest of the New York Polyclinic. The system of Walshe
has been largely followed, with modifications after Flint, Da Costa,
Loomis, Leaming, and others.



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THORACIC DISEASES.

PRINCIPLES OF PHYSICAL DIAGNOSIS.

REGIONAL ANATOMY OF THE CHEST.

A knowledge of Thoracic Diseases is to be obtained mainly by physical diagnosis.

Before entering upon the study of physical signs, it is essential to have thorough knowledge of the structure of the chest, its walls, and the situation and size of the organs in health—that is the regional anatomy or topography of the chest.

TOPOGRAPHY OF THORAX.

REGIONS OF THE CHEST.



1. Anterior Regions.

- 1. Supraclavicular.
- 2. Clavicular.
- 3. Infraclavicular
- 4. Mammary.
- 5. Inframammary
- 6. Suprasternal.
- 7. Upper sternal.
- 8. Lower sternal.

2. Lateral Regions.

- 1. Axillary.
- 2. Infra-axillary.

3. Posterior Regions.

- 1. Upper scapular.
- 2. Lower scapular.
- 3. Infrascapular.
- 4. Interscapular.

ANTERIOR REGIONS.

1. Supraclavicular.

Boundaries.

(a.) Internal . . . Trachea.

(b.) Superior and external, Line drawn from upper part of trachea to acromion process.

(c.) Inferior, . . . Upper border of clavicle.

It is a triangular space,

THE STRUCTURES CONTAINED ARE :

(1) Apex of lung.

(4) Subclavian artery.

(2) Carotid artery.

(5) Subclavian vein.

(3) Jugular vein.

2. Clavicular.

Location, behind the inner three-fifths of the clavicle.

THE STRUCTURES CONTAINED ARE :

(A.) On right side. (B.) On left side.

1. Lung tissue.

1. Carotid artery.

2. Subclavian artery.

2. Lung tissue.

3. Arteria innominata.

3. Subclavian artery.

3. Infraclavicular.

Boundaries.

(a.) Internal, . . . Edge of sternum.

(b.) Superior, . . . Lower border of clavicle.

(c.) External, . . . A line falling vertically from junction of middle and outer third of clavicle.

(d.) Inferior, . . . Lower border of third rib.

THE STRUCTURES CONTAINED ARE :

(A.) On right side. (B.) On left side.

1. Superior lobe of lung. 1. Superior lobe of lung.

- | | |
|--|--|
| 2. Main bronchi, behind second costal cartilage. | 2. Main bronchi, a little below the second costal cartilage. |
| 3. Descending vena cava. | 3. A portion of pulmonary artery immediately behind the second sterno-costal articulation. |
| 4. Portion of arch of aorta, behind second costo-sternal articulation. | 4. Base of heart at lower boundary. |

4. Mammary.

Boundaries.

- (a.) Internal, Edge of sternum.
 (b.) Superior, Lower border of third rib.
 (c.) External, A vertical line continuous with the outer boundary of third region.
 (d.) Inferior, Lower border of sixth rib.

On the right side in this region the thin sharp border of the lung very nearly corresponds to the inferior boundary. But below the lower border of fifth rib the thin portion of lung overlies the liver. The liver is sometimes pushed up into fourth intercostal space.

On the left side a section is taken out of lung to afford space for the heart. The lung extends in front as far as the fourth sterno-costal articulation, where the anterior border passes outward to the outer end of the fifth cartilage, then obliquely inward to lower border of outer end of the sixth costal cartilage, then outward.

STRUCTURES WITHIN THE MAMMARY REGION.

(A.) On right side. (B.) On left side.

- | | |
|-----------|------------------|
| 1. Lung. | 1. Lung. |
| 2. Liver. | 2. Left auricle. |

3. Right auricle and right superior angle of right ventricle close to sternum, between third and fifth ribs.

4. Portion of apex of right ventricle, in fifth intercostal space, one inch within and two inches below nipple.

(a.) Nipple varies somewhat in position. May be said to be, usually, on the fourth rib, at junction of costal cartilage and rib, or in fourth intercostal space.

(b.) Fissure between upper and middle lobes of right lung passes obliquely upward and backwards from fourth cartilage; fissure between middle and lower lobes, upwards and backwards from fifth inter-space; fissure between lobes of left lung begins at fifth inter-space, below the nipple.

5. Inframammary. *Boundaries.*

- (a.) Superior, . . . Lower border of sixth rib.
 (b.) Inferior, . . . Curved line corresponding to the free border of ribs.
 (c.) Internal, . . . Inferior portion of sternum.
 (d.) External, . . . The continuation of the external boundary of mammary region.

STRUCTURES WITHIN THE INFRAMAMMARY REGION.

(A.) On right side. (B.) On left side.

- | | |
|------------------------------|-----------------------------------|
| 1. Lung on full inspiration. | 1. Portion of left lobe of liver. |
| 2. Liver. | 2. Stomach. |
| | 3. Portion of spleen, sometimes. |

6. Suprasternal.

This region is the hollow space immediately above the notch of the sternum, and bounded on either side by the sterno-cleido mastoid muscle.

STRUCTURES WITHIN THE SUPRASTERNAL REGION.

1. Trachea.
2. Arteria innominata at lower right angle.
3. Arch of aorta sometimes reaches to its lower border, where it may often be felt, on firm pressure downward.

7. Upper sternal.

This region includes the space behind the sternum from the notch to the lower border of the third rib.

STRUCTURES WITHIN THE UPPER STERNAL REGION.

1. Lung in front.
2. Bifurcation of trachea at upper border of second rib.
3. Ascending aorta at lower end of region.
4. Transverse aorta, crossing on level with second rib.
5. Innominate artery, at junction of second right costal cartilage.
6. Portion of right auricle in lower part.
7. Aortic valves, midway between centre of sternum and lower edge of left third costal cartilage.
8. Pulmonary valves, one half inch higher than the aortic, on left edge of sternum.

The position of the lung in mid-sternum varies somewhat during inspiration and expiration.

8. Lower sternal.

This region corresponds to that portion of the sternum which lies below the lower border of the third rib.

STRUCTURES WITHIN THE LOWER STERNAL REGION.

1. Portion of body of lung on right side in front, on the left side also as far as the fourth sterno-costal articulation.

2. Greater part of right ventricle.
3. Smaller part of left ventricle.
4. The mitral valves, in the upper part of this region, that is just below the level of the third rib or on a level with the fourth rib, situated close to the left edge of the sternum.
5. The tricuspid valves, situated at about the same level as the mitral, but to the right, nearer to the median line and more superficial.
6. Attachment of heart to diaphragm.
7. Small portion of liver.
8. Portion of stomach, sometimes.

LATERAL REGIONS.

I. Axillary.

Boundaries.

- (a.) Superior, The axilla.
- (b.) Inferior, A line carried backward from the lower boundary of mammary region (sixth rib) to inferior angle of scapula.
- (c.) Interior, The vertical line which forms the common external boundary of the anterior regions.
- (d.) Posterior, The external border of scapula.

This region corresponds laterally with the mammary in front and contains on both sides lung substance, with the main bronchi, deeply seated.

2. Infra-axillary.

Boundaries.

- (a.) Superior, Inferior boundary of axillary region.
- (b.) Inferior, Edges of false ribs.
- (c.) Anterior, Common vertical line, external boundary of inframammary.
- (d.) Posterior, External boundary of infra-scapular region.

This region corresponds laterally to the inframammary and contains on both sides the lower edges of the lung, sloping downward and backward. On the right side is the liver; on the left the stomach and spleen.

POSTERIOR REGIONS.

1. Suprascapular. *Boundaries.*

- (a.) Superior, Second rib.
- (b.) Inferior, Spine of scapula
- (c.) Internal, Internal border of scapula.
- (d.) External, External border of scapula.

Occupied by lung substance.

2. Lower scapular. *Boundaries.*

- (a.) Superior, Spine of scapula.
- (b.) Inferior, Inferior angle of scapula.
- (c.) Internal, Internal border of scapula.
- (d.) External, External border of scapula.

Occupied by lung substance.

3. Infrascapular. *Boundaries.*

- (a.) Superior, Inferior angle of scapula and seventh dorsal vertebræ.
- (b.) Inferior, The twelfth rib.
- (c.) Internal, The spinous processes of the vertebræ.
- (d.) External, Posterior boundary of the infra-axillary.

STRUCTURES WITHIN SUPRA-SCAPULAR REGION.

(A.) On right side. (B.) On left side.

- 1. Lung, to the eleventh rib. 1. Lung, to eleventh rib.

- | | |
|--|---|
| 2. Liver, immediately underneath the surface, between eleventh and twelfth ribs. | 2. Intestines, internally. |
| 3. Small portion of kidney. | 3. Spleen, externally. |
| | 4. Descending aorta, close to the spine. |
| | 5. Portion of the kidney, more than on right side close to spine. |

4. Interscapular.

Boundaries.

- (a.) Superior, . . . The second rib.
 (b.) Inferior, . . . The sixth rib.
 (c.) Internal, . . . Spinous processes of the dorsal vertebræ.
 (d.) External, . . . Inner border of scapula.

This region contains, on both sides, lung substance, the main bronchi, and the bronchial glands. Opposite the third dorsal vertebræ (the second rib in front), is the bifurcation of the trachea. It also contains on the left side the æsophagus, and from the upper part of the fourth dorsal vertebræ downward, the descending aorta.

Useful signs are few. From the second to the sixth rib behind, sounds of air in the trachea and main bronchial tubes are heard. At the seventh or eighth dorsal vertebræ on the left side is the point corresponding to the situation of the mitral valve. It is in this neighborhood that mitral murmurs are readily heard. They may also be heard as high up as the fifth dorsal.

METHODS OF PHYSICAL DIAGNOSIS.

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|-----------------|--|
| 1. Inspection. | 5. Auscultation. |
| 2. Mensuration. | 6. Auscultatory Percussion. |
| 3. Palpation. | 7. Succussion. |
| 4. Percussion. | 8. Determining situation and size of surrounding organs. |

INSPECTION

Is the actual observation of a person's chest. As a means of diagnosis it is secondary in importance to auscultation and percussion, yet is of considerable value. By inspection we recognize the form, size, and general movements of the chest. In order to recognize changes of disease it is necessary to have exact knowledge of the features presented in health.

FORM.

The natural form of a normal chest is a cone with apex upward. Let us look at the different regions of the chest.

The supraclavicular region in health is slightly concave, The suprasternal region is always a deep hollow. The infraclavicular slightly convex. The upper sternal is about level. The lower sternal is always somewhat hollow. The mammary and inframammary are rotund. The intercostal spaces are always slightly concave in both inspiration and expiration, unless there be a superabundance of adipose tissue.

The two sides are very nearly symmetrical. It is estimated that not over twenty per cent. of people have a perfectly symmetrical chest. The most common deviations from a perfect form are slight curvatures of the spinal column. Slight modifications are consistent with health. The main point is to see if the volume of the two sides is about equal.

MOVEMENTS.

1. THORACIC MOVEMENTS.—In inspection, we observe the sternum moves forward, all the ribs except the lower four go upward and outward. The lower four move outward directly, or outward and downward. The ribs all separate from each other in inspiration. In expiration, reverse movements.

2. ABDOMINAL MOVEMENTS.—Diaphragm ascends, and abdomen falls, in expiration ; reverse, in inspiration.

DEPARTURES FROM HEALTH

- | | |
|----------------|-----------------|
| 1. Expansion. | 5. Procidentia. |
| 2. Bulging. | 6. Elevation. |
| 3. Retraction. | 7. Curvature. |
| 4. Depression. | 8. Distortion. |

EXPANSION AND BULGING.

By expansion we mean a general enlargement of one side or of the whole chest ; by bulging, a partial enlargement at one point—enlargement due to causes within the chest. In Asthma and general vesicular Emphysema we have an example of expansion. The shoulders are raised, all the muscles of respiration are in a state of tension, that is, all the muscles in upper part of thorax. The chest is enlarged in every way.

Ordinary Emphysema from obstructive causes produces bulging in the supra and infraclavicular regions. In pleuritis, bulging occurs in the mammary and axillary regions after effusion has taken place. Enlarged heart causes bulging in precordial region. Bulging in inframammary region on right side results from enlarged liver, on the left side from enlarged spleen.

RETRACTION AND DEPRESSION.

Retraction implies general reduction of the whole chest or of one side ; depression has reference to a more local drawing in of the chest wall.

Pleuritic effusion occurs, filling up the pleural cavity so that the lung is compressed, and becomes bound down by inflammatory adhesions. After the fluid is absorbed the lung does not again expand to its previous dimensions. In this case atmospheric pressure acts upon the external surface, causing a retraction of the side.

Depression occurs from a cavity in the lung, from true croup, acute or chronic laryngitis, and from atelectasis, and local pleuretic adhesion.

Expansion and bulging are due to internal and not to external causes. Retraction and depression are due to the co-operation of an external cause, namely : pressure of the atmosphere.

PROCIDENTIA AND ELEVATION.

These terms may apply to changes either in the whole or a part of the chest.

PROCIDENTIA.—Falling of the shoulders and lungs lower than natural, over atrophied or contracted lungs.

ELEVATION.—The lungs fixed higher than normal, or raised in the inspiratory act, as in emphysema.

CURVATURE AND DISTORTION.

1. Lateral curvature of the spine is frequently the result of contraction of one lung or of chronic changes in the pleura with enfeeblement of the respiratory muscles. Angular curvature is associated with early malnutrition, and the existence of chronic bronchitis, asthma, and emphysema.

2. The most common form of distortion is the "pigeon-breast." This is constituted by a flattening of chest laterally, and an arching forward of the sternum. Causes : rachitis, atelectasis or non-expansion of the bronchial tubes in early life, whooping cough, croup, and bronchitis.

The deformity is not due wholly to rachitis, but to the partial, and uneven filling of the bronchial tubes, to laryngeal, or bronchial constriction and co-operation with atmospheric pressure.

PALPATION.

Palpation, the act of laying on the hand. It is less useful than inspection, in ascertaining general size, form, and movement, but is useful in determining local expansion, and the character of vibrations.

The palmar surface of the hand should be laid gently and evenly on the surface. If the hand be applied to the surface of the chest while a person is speaking, a tremulous vibration will be felt. If the individual coughs a vibration is perceived. Bronchitic rales, pleuritic friction, and aneurismal thrill will also give rise to a vibration or fremitus. Hence we have,

- 1.—Respiratory fremitus—Produced by breathing.
2. Vocal fremitus—Produced by the voice.
3. Tussal fremitus—Produced by cough.
4. Bronchial fremitus—Produced by bronchitis.
5. Pulsatile fremitus—Produced by aneurism.
6. Friction fremitus—Produced by pleurisy.

Ordinarily when we use the term fremitus we mean vocal fremitus.

Palpation may also detect fluctuation. In pneumo-hydrothorax, or in a very large cavity where air passes in and out, the sensation of fluctuation may be felt by the hand. Such fluid is more often detected by succussion. In order to have succussion there must be both fluid and air in the cavity.

RESPIRATION.—The frequency and rhythm of respiration are also noted by palpation. The average in the adult is eighteen to twenty respirations per minute. Having

counted the respirations, put one hand on the pulse and count pulsations, noting the relation between the two.

Normally the ratio of respiration to pulse is as one to four. It takes four times as long for the blood to go through the systemic as through the pulmonic circulation.

In disease the pulse-respiration ratio is subject to much variation. In chorea it has been observed to be—

Pulse : respiration : : 9 : 1, and in hysteria, pulse : respiration : : 5 : 1.

MENSURATION.

There are many measurements, but the principal is the circular, taken on the level of the sixth costal cartilage. The average circular chest measurement of a healthy adult male, five feet eight inches in height, midway between inspiration and expiration, is 33 inches. It may go as high as 44, or as low as 27 inches. The average is a little less for women.

DIFFERENCE OF RESPIRATION OF MEN AND WOMEN.

Respiration in the male is mostly abdominal, in the female mostly thoracic. This is in part owing to dress, but chiefly to sex, as women in pregnancy are obliged to use the thoracic muscles in respiration.

CIRCULAR EXPANSION.

In ordinary quiet respiration, the circular expansion varies from one-fourth or one-half an inch to one inch. It may be as high as two inches. If a person takes a forced expiration, and the chest be measured, and then a full inspiration and the measurement be taken, the difference will represent the full expansive power. It varies from two and one-half to three and one half inches, but may be more. The expansive power for women should be about the same, though their stature is less.

QUANTITIES OF AIR.

The capacity for air is measured by a spirometer. The air habitually in the lungs, or capable of being introduced into them by respiration, is divisible into various quantities:

- | | |
|-----------------------|-----------------------|
| 1. Persistent air. | 4. Tidal air. |
| 2. Residual air. | 5. Complementary air. |
| 3. Supplementary air. | |

DEFINITIONS.

1. *Persistent air* is that which cannot be removed from the lung structure by any possibility, not even by heavy pressure after death. It is removed by disease.

2. *Residual air* is that which is never expired while the thoracic wall is intact, but which escapes when the chest wall is opened on the cadaver, and atmospheric pressure collapses the lungs.

3. *Supplementary air*, that which is not expired in ordinary breathing, but may be ejected by a forcible expiration.

4. *Tidal air* is that which goes in and out in ordinary respiration.

5. *Complementary air* is that which may be inhaled by a forced inspiration after an ordinary inspiration.

The first three Dr. Walshe calls *stagnant air*; the last three represent the total breathing volume and are the only quantities which can be measured by the spirometer. The average amount of tidal air for a healthy adult male is 26 cubic inches for each inspiration. The greatest amount which the average male can inspire is 174 cubic inches. Thus it is evident we do not use one-half our lungs, even in health.

RELATIVE LENGTH OF INSPIRATION AND EXPIRATION.

- Inspiration, 5.
Expiration, 4.
Rest, 1.

This relation is changed in disease. In advanced emphysema expiration is two or three times as long as inspiration.

CHARACTER OF THE TWO ACTS.

Inspiration begins gently, increases gradually in intensity, till it reaches the middle of the act, then gradually dies away, and is immediately followed by expiration.

Expiration begins actively, instantaneously, gently passing into rest.

PERCUSSION.

Percussion is the method of eliciting sounds from the chest in health and disease, by striking the chest wall with the finger tips or the percussing hammer. We have :

1. Immediate percussion.
2. Mediate percussion.

IMMEDIATE PERCUSSION

Is the direct striking of the surface of the chest with the hand or fingers. This is seldom practiced.

MEDIATE PERCUSSION

Consists in the interposition of some media between the striking hammer or fingers and the chest. This is by far the most common mode of percussion. It was rendered a science by Piorry and Laennec. When the hammer is used the interposed medium is a plate of rubber or ivory, and is called a pleximeter. The hammer and pleximeter are useful for clinical teaching, to elicit very clear sounds and for uniformity of stroke. It is better in practice to rely upon the fingers.

MODE OF PERCUSSING.

One finger of the left hand is used as a pleximeter or medium, and one or more fingers of the right hand as a

hammer. The palmar surface of the interposed finger should be placed in accurate and firm contact with the surface. Percussion with one finger may be performed by moving it at the metacarpo-phalangeal articulation (the knuckle). Such a method, however, requires much practice to perfect it, and the stroke will bring out only delicate sounds.

The rule is to strike from the wrist, without moving elbow or shoulder. We thus describe an arc of a circle and bring the finger at right angles upon the surface. The blow should be quick and sharp, and the finger instantly removed, in order to let the chest vibration continue. When percussing over cavities, and not desiring the sound to reverberate, the finger may be retained in contact. A single percussion stroke over a given point is better than repeated strokes.

PERCUSSION DEVELOPS

Sounds indicative of :

1. The degree of elasticity or tension of the chest wall.
2. The condition of the contained organs.

PROPERTIES OF THE PERCUSSION SOUND.

1. *Duration*, the length of sound as regards time.
2. *Intensity*, resonance, amplitude, or volume of sound.
3. *Pitch*, referring to length of sound wave, the acuteness, dullness or flatness of sound.
4. *Quality*, what the sound suggests as to the condition of organs underneath.

(1) DURATION.

The duration of the percussion sound varies perceptibly in different parts of the chest. It is longer, for instance, over the lung substance than over the heart or liver.

A certain relation exists between the four properties of sound : duration, resonance, pitch and quality.

With high pitch there is diminished resonance, hard quality and brief duration. With low pitch, more resonance, softer quality and larger duration.

(2) INTENSITY OR RESONANCE.

The resonance is not due wholly to the underlying organ. The sound produced by percussing over the lungs differs from that caused by percussing over the liver. The difference is due, in part, to the proper tissue of the organ; in part, that the lungs contain air which modifies the sound, and in part, to the position in the chest and relation to other organs. Again, the sound over the heart and the liver, both solid organs, is slightly different; that which the heart gives out is more intense, of longer duration and lower pitch than that over the liver.

Resonance, therefore, is due to :

- (a.) Consistency of the organ.
- (b.) Its contents—air or fluid.
- (c.) The shape and elasticity of the walls encasing it.

(3) PITCH

Refers to the length of the sound wave. We define it ordinarily by saying it is the acuteness or dullness of the sound. Over anything solid, as the heart or liver, we get high pitch; over anything less solid, as over the normal pulmonary tissue, we get low pitch.

The greater the quantity of air the lower the pitch. Pitch is highest over fluids, as over pleuritic effusion, because fluids are most dense. Over gases, pitch is very low indeed. In percussing we find pitch high over the liver, heart and muscle; low over lung tissue, and over stomach lower still.

Pitch frequently has connection with duration. When the pitch is high, the duration is short; when the pitch is low, the duration is long.

(4) QUALITY.

Quality or timbre suggests the nature of the subjacent or underlying organs. We have hardness, softness, and hollowness. The hollow type occurs under three varieties, the tubular, amphoric, and cracked metal.

The *Hard* quality is wooden, resembling that yielded by mediate percussion on a table. It follows percussion over the solid viscera, or over an infiltrated or solidified lung.

The *Soft* quality is that yielded by percussing over normal pulmonary tissue and has been likened to the sound of the drum covered with a coarse woolen cloth.

The *Tubular* quality is that produced by air in an elongated defined cavity. This quality is to be detected over the trachea, and over the primary bronchi.

When anything solid lies between the large bronchi and the costal surfaces, this sound is sometimes obtained ; thus it may occur in the interscapular region from enlarged bronchial glands, over a great part of the back in cases of intrathoracic tumor.

Small excavations in the lung may, and dilatation of the bronchi will, produce this quality of percussion note.

Amphoric quality is the tubular on a large scale. It resembles the sound produced by tapping the cheek when the mouth is closed, and fully but not tensely inflated. The common source of this sound is a cavity of large size near the surface, and provided with hard and thin walls.

Cracked Metal.—Cracked pot or cracked metal quality is an air impulse sound—air in a cavity rushing against its walls. The amphoric quality is almost always coupled with this quality. In the same patient, percussion with mouth and nostrils closed produce amphoric sound, and percussion with the mouth and nose open will produce cracked pot sound. It resembles the sound resulting from striking the back of the hands, loosely folded across each other, against the knee, the contained air being forced out quickly between the fingers

at each blow. If the chest of a crying infant be percussed in expiration the resonance will be of cracked metal quality, and it can be produced by percussion on the elastic chest wall of most young persons.

Tympanitic quality results from an unusual amount of air contained within a cavity with elastic walls. The sound is drum-like. Typical tympanitic sound exists in pneumothorax, and over stomach, or intestines distended by flatus.

The sounds developed by percussion are not always dependent on an organ lying immediately beneath. Sound may be conveyed across the chest. It must be remembered the whole chest is an acoustic box—all the contained organs modifying the sounds.

MUSCLES.

The percussion note of muscles is non-resonant, high, hard, and brief as elicited by percussing the muscles of the shoulder or arm.

HEART.

The heart is only a great muscle, hence its percussion note (superficial area) is high-pitched and hard. These qualities are less marked where it is over-lapped by the lung (deep area). So we have superficial and deep sounds over an organ.

LUNGS.

The breathing surface of the lung is formed by the dividing bronchi and bronchioles and air vessels, with elastic or connective tissue to hold them together. The normal properties of percussion sound differ somewhat over different places.

In the infraclavicular region the resonance is considerable, the quality is true pulmonary—pitch low—duration distinctly perceptible. In the right mammary region the pitch rises some, even at the upper part, and is very perceptibly elevated at and below the fourth intercostal space, owing to

the contiguity of that hard, solid organ—the liver (law of consonance). On the left side, the heart likewise modifies the sound.

The resonance of the right inframammary region, in consequence of the presence of the liver beneath the surface, is high in pitch, brief in duration, hard, almost wooden in quality.

The suprasternal region gives tubular resonance. In the upper sternal region the sound is somewhat of a mixed character as also in the lower sternal region. Here the stomach may further modify the sound. It is in this region that difficulty is often encountered in tracing the line of demarcation between the liver and the heart. The diaphragm intervenes between the two organs, but the percussion note of one differs so little from that of the other that it is often practically impossible to define the line of union.

A good rule in that case is to draw a line from the point of the heart's apex beat to the apex of the angle formed by the union of the upper free edge of the liver with the right free edge of the heart.

Over trachea and large bronchial tubes the percussion sound is tubular, the resonance full, owing partly to the contained air and partly to the walls themselves of the tubes, the pitch is low, not hollow nor tympanitic, and the duration is long and ringing.

Over the aorta, gentle percussion gives true pulmonary percussion note; deep percussion lessens the duration and resonance, raises the pitch and makes the quality hard. If an aneurismal tumor exists the same modifications will be present over a larger area and intensified.

The posterior thoracic regions do not give the pulmonary sounds so well as the anterior regions, because of the thickness of the dorsal muscles. When the scapulæ are carried

outward by the second position of Corson (see below), the interscapular areas are nearly and sometimes more than doubled, and the muscles being rendered tense and thin, a normal pulmonary percussion note is elicited, which continues as we descend and percuss the infrascapular regions. Below the eleventh rib of right side the note is high pitched, hard, hepatic. Resonance does not extend as far down on the right side as on the left. On the left side of the chest, posteriorly, below the eleventh rib, the spleen, if large, or the stomach and intestines may modify the percussion note.

The lateral regions of the chest are more highly resonant. On the right side the percussion sound loses in volume and rises in pitch as we go downwards toward the liver. Below the sixth rib on the left side the sound is modified by the spleen and stomach.

The sound over the stomach is tympanitic, and over the intestines, according as they are empty, full of fæces, or distended with gas, varies from tonelessness, to tympanitic resonance.

The spleen gives the same hard, toneless, hepatic, quality of sound that the liver does.

POSITIONS OF PATIENT FOR PERCUSSION. (*Dr. Corson.*)

First Position (for Anterior Examination).

Shoulders well thrown back; arms folded behind the back, to make tense the muscles on the front of the chest.

Second Position (for Posterior Examination).

Body bent slightly forward; head bowed; arms crossed in front of chest. (Better still to clasp each shoulder with opposite hand.)

Third Position (for Lateral Examination.)

Arms raised vertically upwards; hands folded across top of head.

AUSCULTATION.

Auscultation means the application of the ear to the chest for the detection of normal and abnormal sounds, for, as relates to the respiratory organs, we may hear

1. Normal breath sounds.
2. Normal voice sounds.
3. Abnormal breath sounds.
4. Abnormal voice sounds.
5. New sounds.
 - (a.) Sounds resulting from a combination of above.
 - (b.) Sounds having no connection with either voice or breath sounds.

We distinguish in these sounds the following properties :

- | | |
|---------------|-------------|
| 1. Duration. | 4. Quality. |
| 2. Intensity. | 5. Rhythm. |
| 3. Pitch. | |

THESE PROPERTIES DEFINED.

1. *Duration*, the period or persistence of the sound, usually proportionate to intensity.

2. *Intensity* is the degree of resonance, the volume, or amplitude of the sound.

3. *Pitch* refers to the acuteness of sound, whether the sound is high or low.

4. *Quality* is that property which reveals something of the character of the substance, emitting or conducting the sound.

Rhythm refers to the regular recurrence of sounds, whether uniform or irregular and jerking.

METHODS OF AUSCULTATION.

1. *Immediate*, if the ear is applied to the chest.
2. *Mediate*, if an instrument be used.

THE STETHOSCOPE.

The instrument used in mediate auscultation is a stethoscope. The original stethoscope of Laennec was a hollow

cylinder of wood, having an expanded end, its longitudinal axis being parallel with the grain of the wood.

Stethoscopes have also been made of other materials, as glass, gutta-percha, metal, and ivory, but wood is preferable.

Dr. Cammann's is a double flexible stethoscope, having two ear pieces, uniting in a single tube. Scott Alison's has two distinct tubes united by a joint. They pass one from each side of the chest to either ear of the listener. The advantage claimed is the facility afforded in comparing the sounds from the two sides. Practically there is no advantage. The mind, in attempting to take cognizance of the sounds on two sides, is confused.

Dr. Cammann's stethoscope is used very largely in this country ; its use is discouraged by English and continental writers. Their criticism is that it not only intensifies sounds but changes them, altering their pitch and quality.

Snelling's soft rubber cap is employed on Cammann's stethoscope, in examining emaciated patients.

Both immediate and mediate auscultation have their advantages.

ARGUMENTS IN FAVOR OF THE USE OF THE STETHOSCOPE, OR MEDIATE AUSCULTATION.

1. It localizes a sound, as when produced in a small cavity, or a heart murmur.
2. The delicacy of ladies is favored. It obviates chest exposure.
3. It avoids contact of the head with the bodies and clothes of uncleanly patients.
4. Certain, otherwise not very distinct, sounds are by the stethoscope rendered more intense.

ARGUMENTS AGAINST THE USE OF THE STETHOSCOPE AND FAVORING DEPENDENCE ON THE EAR ALONE.

1. The interposition of a solid material may intensify the sound at the same time that it modifies or changes it. This is especially true of the double stethoscope.

2. A person using the stethoscope as a clinical teacher, or in practice, may lose the delicacy of his ear.

AUSCULTATION IN HEALTH.

Putting the ear to the chest we listen, first, to the respiration. In one-fourth of healthy persons inspiration alone is heard ; in one-fourth of healthy persons expiration is wanting.

Where inspiration and expiration are both heard, inspiration is from three to four times as long as expiration, that is, as they are heard. (Inspiration as a physical act is five-fourths as long as expiration.)

RATIO OF INSPIRATION TO EXPIRATION.

As physical act :

Inspiration.....	5
Expiration.....	4
Rest.....	1

Ratio of inspiratory to expiratory sound, as heard over chest wall :

Inspiration.....	3 or 4
Expiration.....	1

Inspiration is heard to be a soft, breezy sound, neither moist nor dry, which is faint at its inception, gradually developed, increasing in intensity and volume to its mid-period and gradually subsides. Its rhythm is steady, uniform and continuous.

The expiratory sound is harsher, more hollow, and only one-third or one-fourth as long or as loud. Inspiration is an active, expiration a passive movement. In inspiration the chest walls are raised and expanded by the contraction of muscles—the abdomen distends from the pressure downwards of the diaphragm.

In expiration there is a movement of the abdominal viscera upward, and subsidence of the bony structures of the thorax.

Inspiratory sound is heard, and is long :

1. Because the currents of inspired air are entering a system of tubes steadily decreasing in size, and developing air-and-tube friction.

2. The currents of air come towards the chest wall, and bring the sound towards the listener's ear.

Expiratory sound is faintly heard and short:

1. Because the expiratory currents are passing out of a system of tubes, steadily increasing in size. Hence but little air-and-tube friction is developed.

2. The currents of expired air and the sounds which they create are receding from the chest wall and away from the listener's ear.

The last statement may be verified by listening with either the ear or the stethoscope at a person's mouth. Expiratory sound is found to be there quite as distinct and as long as inspiratory sound.

Various theories have been advanced to account for the respiratory sounds.

FACTORS OF NORMAL SOUNDS OF RESPIRATION.

- | | |
|----------------|------------------------|
| 1. Pharyngeal. | 5. Vesicular. |
| 2. Laryngeal. | 6. Parenchyma of lung. |
| 3. Tracheal. | 7. Muscular sussurus. |
| 4. Bronchial. | |

The respiratory sounds have certain elements enumerated in the list above. The pharyngeal and laryngeal elements are not heard except when there are some changes, as enlarged tonsils, laryngitis, etc. We do not hear them normally because they are slight, and not observed except when intensified by some obstruction. In inspiration, the air comes against the sides and bifurcations of the trachea and bronchial tubes.

The bronchial element of respiratory sound is thus produced. Its character is tubular—it has an air-and-tube friction quality.

Vesicular breathing is a breezy, fine continuous sound, at least continuous in inspiration. There are many and opposing theories as to the cause of the normal vesicular sound. Some claim that air passing through the bronchial tubes produces an air-friction sound, and in passing through the ultimate bronchial tubes, by dilatation of their walls produces the vesicular murmur.

According to the views of others, as the air leaves the ultimate bronchial tubes, which are more or less rigid, and which terminate in the more expanded and elastic infundibuli, the change or release from the confined tubes to the air spaces is the cause of the sound termed vesicular.

MUSCULAR SUSSURUS.

The muscular action going on in the chest wall gives rise in some persons to a peculiar buzzing sound, termed muscular sussurus ; its amount does not seem to be directly as the muscularity of the individual. It is increased by efforts of all kinds, as for instance, that of maintaining an uncomfortable posture ; it is continuous not synchronous with respiration, and rather increases than diminishes in intensity when the breath is held. It may in some places be suspended by putting the muscles in a state of relaxation, but in the infra-axillary regions, where it is sometimes highly marked, it cannot be thus arrested.

SEX.

Women breath largely by the upper part of the chest ; men more by the lower part. We are more likely to hear the expiration of women, it is harsher in character.

CHILDREN.

The tubular or bronchial element is more marked in children, and both inspiration and expiration may be intense and harsh—designated “peurile breathing.” Expiration will also be heard during its entire period. Such sounds in an adult would denote disease.

VOCAL RESONANCE.

In health we say little about the auscultation of the voice ; yet if we put our ear to a healthy chest while the person is speaking we perceive a "vocal resonance," not distinct words. The sound is vague and distant. In consolidation of the lung from any cause, as exudation in the second stage of pneumonia, or a deposit of tubercle or cancer in the lung, the voice is brought directly to the ear. This constitutes bronchial voice or bronchophony (see later), and replaces vocal resonance.

The vocal resonance of males is greater than that of females ; the deeper the voice, bass or baritone, the greater the resonance.

CHEST ACOUSTICS.

- | | | |
|----------------------------------|---|---|
| 1. <i>Unison resonance.</i> | } | Methods
of development of
sounds. |
| Reinforcement by thoracic walls. | | |
| 2. <i>Consonance.</i> | | |
| Reinforcement by other organs. | | |
| 3. <i>Echo.</i> | | |

(1.) UNISON RESONANCE.

By unison resonance is meant the reinforcement of sound by the thoracic walls, as occurs in the box of a guitar or violin, when notes are produced from their strings, or as when a music box instead of being held in the air is placed on a table. There can be little doubt but that in the natural condition of the chest the principle of unison resonance is illustrated.

(2.) CONSONANCE.

By consonance in music is meant the reproduction of certain notes of instruments or of the voice by other instruments standing near. It is difficult to prove that there is in the chest a repetition of sound by consonance, but contiguous structures exert a modifying influence, notably the liver, stomach, and intestines, upon the chest sounds.

(3.) ECHO.

Repetition of sounds. The echo is not heard over the vesicles, but in a cavity, as in phthisis or pneumothorax.

AUSCULTATION IN DISEASE.

In disease we study changes from normal conditions.
Respiratory sounds :

1. Changes of duration and intensity:

(a.) Exaggerated.	(c.) Suppressed.
(b.) Weak.	
2. Changes in rhythm:

(a.) Jerking.	(d.) Unfinished.
(b.) Divided.	(e.) Altered ratio of inspira-
(c.) Deferred.	tion to expiration.
3. Changes in quality, pitch and rhythm combined :

(a.) Harsh.	(b.) Blowing.
Rude.	Tubular.
"Broncho-vesicular."	Cavernous.
Bronchial.	Amphoric.

CHANGES IN DURATION AND INTENSITY.

Sounds may be exaggerated, weak or suppressed. Exaggerated in bronchitis. In convalescence quality may be normal and intensity weak. Suppressed—an absence of respiratory sounds.

CHANGES IN RHYTHM.

(a.) Jerking.—When the movement of inspiration instead of being accompanied by a continuous sound, is attended with an interrupted one, divided into several unequal parts, the respiration is called jerking. Illustrated in asthma, from obstruction in tubes, in early stage of pleurisy, pleurodynia,

intercostal neuralgia, hemiplegia, and spinal irritation. In some cases of incipient tubercle, the rhythm of sound is disturbed.

(*b.*) Divided.—When the heart's action is such as to throw the blood so violently into the lung as to cut the act of respiration into four or five parts, the character is remarked.

(*c.*) Deferred inspiration.—When the inspiratory action commences and continues for a short time before sound is produced, it is said to be deferred. This is remarked in emphysema.

(*d.*) Unfinished inspiration.—In certain cases the inspiratory sound ceases before the chest expansion has been completed. This occurs only over exceptional cases of consolidation.

(*e.*) The altered ratio of inspiration and expiration is to be noticed.

In health, expiration is one-third or one-fourth of inspiration. In emphysema, expiration is greatly prolonged so that it may be four times as long as inspiration..

Changes in quality, pitch, and rhythm combined.

(*a.*) Harsh.—Both sounds have lost their soft, breezy character, and have become sharper and more blowing in character. Harsh respiration is heard in condensation of the lung to a slight degree, and in the dryness of mucous membrane of bronchi. The sound produced in bronchitis we ordinarily understand as tube friction sound.

(*b.*) Blowing sounds.—Low and long—simple—may be diffused or tubular. Low, as though blowing in a bottle—amphoric. Cavernous sounds differ in degree as to intensity, pitch and quality.

SOUNDS PRODUCED IN BRONCHIAL TUBES.

The most ordinary sounds are those produced in bronchial tubes.

Sounds produced in bronchial tubes and having a dry character are *rhonchi* ; moist sounds are *rales*.

- | | |
|-----------------|-------------------|
| 1. Dry sounds : | 2. Moist sounds : |
| (Rhonchi.) | (Rales.) |
| Sibilant. | Mucous. |
| Sonorous. | Sub-mucous. |
| | Sub-crepitant. |

1. Sibilant and sonorous rhonchi differ only in pitch. The former being very high in pitch, almost hissing, the latter lower and graver. They are produced by air passing through contracted portions of the tubes, and the more reduced the calibre of the tube, the more sibilant character will the sound possess. They are perfectly dry sounds, and have nothing to do with mucus. Irregular contractions in asthma give the greatest variety of sounds.

2. The moist sounds, or rales, are produced in the tubes of moderate or considerable calibre by the breaking of bubbles of air in fluid, either serum, sero-mucus, or mucus. Their distinguishing characteristic is that they disappear or are modified on coughing, the action having dislodged the mucus. They are common in bronchitis, bronchial hemorrhage, pneumorrhagia, and in evacuation of pus into the bronchi.

BRONCHORRHAGIA.—THE CREPITANT RULE.

The terms crepitant and sub-crepitant are reserved for sounds produced in the vesicles and terminal tubes.

There are different theories in regard to the production of these sounds. One theory is that the crepitant rale is produced by air coming down into the air cells and breaking through the exudation matter. Another theory is that the air sacs being collapsed, on inspiration their glutinous walls separate, giving rise to the crepitant rale. The same sound may be simulated by pressing a piece of sponge rubber close to the ear and then letting it relax. In its relaxation it pro-

duces far more sound than in its compression. Another theory is that it is due to stretching of the intervesicular tissue. The inflammatory exudation, it is claimed, starches or stiffens, as it were, the intervesicular tissue, and renders its expansion more difficult and productive of sound.

Crepitant rale is heard only during inspiration, and in two diseases, viz. : croupous pneumonia and oedema of the lung.

THE SUB-CREPITANT RALE.

The sub-crepitant rale is distinguished from the crepitant by being double. It is heard in inspiration and expiration. It is also less fine and more plastic or glutinous in quality. It is produced by minute air currents breaking through mucus in small bronchial tubes. It is heard in "suffocative catarrh," or capillary bronchitis. Sub-crepitant and plastic rales, unless uniformly diffused over both lungs, are quite likely to be of pleural origin.

RECAPITULATION OF CHEST SOUNDS IN DISEASE.

(1.) ABNORMAL SOUNDS PRODUCED IN AIR PASSAGES.

(*Walshe.*)

Whistling.	{	High-pitched—Sibilant.
	{	Low-pitched—Sonorous.
Crepitating.		
Crackling.	{	Dry.
	{	Moist.
Bubbling.	{	Small. { Sub-crepitant.
		{ Sub-mucous.
	{	Large.—Mucus. { Simple.
		{ Hollow.

Bronchial affections are the most numerous of all pulmonary affections, so that their signs, mucous, and sub-mucous rales, sibilant and sonorous rhonchi, are the most common signs heard by the practitioner.

Next to bronchial affections in relation to frequency, are slight and severe attacks of pleurisy.

(2.) ABNORMAL PLEURAL SOUNDS.

(*Walshe.*)

- | | |
|-----------------|------------------------|
| 1. Single. | 2. Jerking—Divided. |
| 1. Faint. | 2. Loud. |
| (a.) Attrition. | { Grazing.
Rubbing. |
| (b.) Creaking. | (d.) Rumbling. |
| (c.) Crackling. | |

In health there is no sound produced by the colliding of the two opposed pleural walls. Their smoothness and moisture allow them to glide noiselessly upon each other. When these conditions have become modified by disease the gliding is accompanied with different modifications of sound.

EXPLANATORY OF PLEURAL SOUNDS.

(*Walshe.*)

1. Single sounds may be of any character, variable in intensity, pitch and duration.

2. More usually the sounds produce a series of abrupt jerking noises, which also may be of variable character and duration, commonly intensified by the act of inspiration, generally heard also in expiration.

Again, these sounds may be (1) Faint or (2) Loud. (a.) Attrition. The first condition in inflammation of the pleura is a removal of the fluid or moisture which lubricates the part. The surfaces are next denuded of epithelium, tumefied and roughened. The movements of these surfaces give rise to a grating, rubbing sound, found entirely in recent cases. If two denuded and villous surfaces have grown together at points, the pulling or stretching of these adhesions may give rise to sub-crepitant or plastic rales.

(*b.*) Creaking sounds are more leathery. They indicate dryness, firmness, and toughness of the inflamed surfaces and exudation matter. Heard in chronic cases.

(*c.*) Crackling sounds are produced when there is moisture in the plastic matter within the pleura.

(*d.*) Rumbling: friction sound prolonged during the two acts of respiration; a series of distinct, interrupted jerks.

The signs of intrapleural pathology have been systematized by Prof. James R. Leaning in this country, and by Sir Andrew Clarke abroad.

SOUNDS OVER SERUM IN PLEURAL CAVITY.

A slight amount of serum masks respiratory and voice sounds, and gives aegophony.

A large amount obscures all sounds—respiratory, vocal, tussal—and may give aegophony at upper border of liquid.

ABNORMAL SOUNDS PRODUCED IN PARENCHYMA OF LUNG.

1. Changes of respiratory sounds.

- (1.) Slight consolidation—feeble or absent vesicular murmur, respiration exaggerated, rude, harsh, “broncho-vesicular.” Inspiration and expiration both raised in pitch and expiration prolonged.
- (2.) Marked consolidation, absence of vesicular murmur, high pitch of both inspiration and expiration, the latter clear and prolonged, bronchial breathing.
- (3.) Cavity—cavernous breathing.

2. Changes of voice sounds.

- (1.) Slight consolidation, diminished vocal resonance, raised pitch of voice, bronchial whisper.
- (2.) Marked consolidation, absence of normal vocal resonance; bronchophony and bronchial whisper.
- (3.) In cavity, if small—cavernous whisper; medium, —cavernous voice; large—pectoriloquy.

3. Changes in cough sound.

Tussal resonance lessened by slight consolidation, and absent in marked consolidation, bronchial cough in consolidation, cough will also be cavernous when excavation is large.

4. New sounds.

Gurgles, mucus rales, creaking in walls of cavities, metallic tinkles in large cavities.

AUSCULTATORY PERCUSSION.

The sixth method of physical exploration consists in definitely locating and determining the size of organs by listening over them with the stethoscope, while percussion is made towards them from all sides. The points of impact are detected by the listener, the moment the border of the organ is reached, by reason of the definite conduction of percussion note. The several points so found are then connected by a line, and the periphery of the organ determined with accuracy. The method is specially applicable to the heart, liver and spleen.

SYNOPSIS OF ACUTE BRONCHITIS OF ADULTS.

Definition.	Inflammation of bronchial tubes.
Pathology.	Sub-mucous hyperæmia : acute catarrhal process. 1st Stage.—Surface dry and tumefied. 2d Stage.—Surface bathed with serum, sero-mucus or muco-pus. Calibre of tubes { Sub-mucous congestive swelling. may be changed by { Local spasm of muscular coat. { Adhesion of viscid mucus.
Causes.	"Taking cold"; chilling surface; inhalation of hot and cold air; irritant vapors; atmospheric causes (influenza). Secondary in specific blood states:—measles, typhoid and typhus fevers, variola, etc.
Symptoms.	May or may not be preceding coryza:— Soreness, rawness, tightness, oppression in upper sternal region, increased by cough; little or no fever in most cases; heavy, deep, sonorous, paroxysmal cough; no dyspnœa; inspiration long and full. Sputa in first stage scanty or absent. In second stage, at first, sputa is serous, transparent, frothy; later, white, viscid, opaque—mucus; later, yellow tenacious, muco-pus. (Mucin and epithelial scales.)
Physical Signs.	Both stages, normal percussion resonance; normal, vesicular sounds by auscultation. First stage—tubular breathing—harsh, exaggerated, sonorous, sibilant rhonchi. Second stage—mucous and sub-mucous rales, of various degrees of liquidity; to and fro gurgles in main bronchi. All removed or modified by cough or expectoration.
Diagnosis.	Normal percussion resonance and vesicular sounds; rhonchi and rales bi-lateral. A symmetrical disease. Absence of chill. "stitch in side," or high fever; successive changes in sputa.
Prognosis.	Good. In adult does not extend to pneumonia, but some cases become chronic. Epidemics more severe—broncho-pneumonia.
Treatment.	Prophylactic—warm clothes, diet, air, exercise. Abortive—opiates, diaphoretics, saline purge. Palliative, anti-spasmodics and expectorants, supporting remedies.

SYNOPSIS OF CAPILLARY BRONCHITIS.

(SUFFOCATIVE CATARRH.)

Definition.	A form occurring in young children. A bronchitis of small tubes. Often an obstruction of small (fine) tubes and collapse of lobules; sometimes extending to vesicles (lobular, catarrhal pneumonia).
Pathology.	Inflammation of smaller bronchi, catarrhal products, choking of tubes, more or less.
Causes.	Predisposing.—Early infancy, great weakness, errors of diet. Exciting.—Colds, measles, etc.
Symptoms.	Labored respiration, exhaustion, imperfect oxygenation, pallor, coolness, carbon dioxide poisoning, little pain or oppression. If any sputa, white small bodies, or tenacious mucus.
Physical Signs.	Percussion normal; auscultation—sub-crepitant rale in inspiration and expiration.
Diagnosis.	Age, labored breathing, asthenia; resonant chest, pathognomonic sub-crepitus (double). If extended to vesicles, crepitant rale. If plugging of bronchus and collapse of lobule, there may be a local point of high pitch and bronchial breathing. If collapse is extensive, retraction of chest on inspiration, and sinking at suprasternal notch and epigastrium.
Prognosis.	Always grave.
Treatment.	Support vigorously by diet, quinine, stimulants, as needed; oil silk jacket; mild counter irritants; warm, moist, uniform air; oxygen. Emesis; if needed to eject mucus.

CHRONIC BRONCHITIS.

Definition.	<p>Acute attack often leaves bronchi liable to frequent sub-acute attacks, which may result ultimately in chronic bronchitis.</p> <p>A variable form of sequelae of previous bronchial catarrhs.</p>
Pathology.	<p>Varying in different cases, including one or more of the following pathological states :</p> <ol style="list-style-type: none"> 1. Simple chronic catarrh or relaxed, thickened mucous surface. 2. Changes of calibre of bronchi. General relaxation or dilatation. Local relaxation. Local contraction or obliteration. (Cirrhosis of lung the result.) 3. Local ulceration of mucous surface. Local ulceration of all the coats. 4. Peri—or extra—bronchial inflammatory deposits. 5. The same broken down, leaving cavity opening into bronchus. 6. Resultant pneumonia, indurations.
Symptoms.	<p>Chronic cough, for months and years, without great loss of flesh, strength or appetite.</p> <p>Chronic catarrh of bronchi ; quantity, color, viscosity, determined by extent and kind of lesion.</p>
Physical Signs.	<p>Varying with simple bronchial, peri-bronchial or pulmonary lesions, with dilated bronchi, bronchial cavity, peri-bronchitis or cirrhosis of lung. Simulate every form of phthisis.</p>
Diagnosis.	<p>To be differentiated from Tuberculosis.</p>
Prognosis.	<p>Persistent, often incurable, but slow progress; remissions and exacerbations.</p>
Treatment.	<p>Hygienic, tonic. Climatic.</p> <p>Palliate symptoms according to case.</p> <p>Balsamic inhalations. Iodide of potassium.</p>

SYNOPSIS OF ASTHMA

Definition.	A functional disease; labored obstructed respiration, due to spasm of bronchi, usually with bronchial catarrh.
Pathology,	Theory of hyperæmia of par vagum. No known lesion of asthma. May be co-existing lesions of acute or chronic bronchitis, emphysema, etc.
Causes.	Often obscure or unknown. May be hereditary. Excited by cold air, dust, vapor, fatigue, mental effort, excitement; especially indigestion and bad ventilation (foul air of sleeping room) flatulence, constipation, uterine disturbance (reflex action). Most common in men. Special form.--"Hay asthma" or autumnal catarrh, due to emanations of flowing, mature vegetation.
Symptoms.	Attack often sudden (rarely gradual, with preceding catarrh). Often at night in sleep. Increasing sense of obstruction to admission of air; suffocation; face anxious; respiratory muscles fixed; orthopnoea; head thrown back; gasping; dilated nares; obstructed venous circulation; in head and neck, symptoms of carbonic acid gas accumulation; fatigue, exhaustion. Sudden relief, or gradual, with lingering bronchial catarrh.
Physical Signs.	Normal pulmonary resonance, or vesiculo-tympanic resonance on percussion. Fine tube and vesicular sounds wanting, or feeble. Expiration long and changing, composed of numerous sibilant and sonorous rhonchi, of varied and changing intensity, pitch, and quality. Moist rales of bronchial catarrh exist.
Diagnosis.	Paroxysms sudden; physical signs, definite recovery from attack, complete and speedy, seldom any permanent dyspnoea.
Prognosis.	May cause emphysema. Remotely effects the heart.
Treatment.	Of paroxysms: Oxygen, chloroform. Hypodermic use of morphia and atropia. Stramonium. Nitre cigarettes. Of intervals: Quinine. Iodide of potassium.

SYNOPSIS OF PLEURITIS.

Definition.	Inflammation of pleura with accumulation of inflammatory products on free surface, or in the pleural cavity.
Pathology.	<p>Hyperæmia of sub-serous vessels ; surface denuded of epithelium; rough, "hazy," later escape of exudation, may be plastic, sero-plastic or wholly serous. If adhesions, they are due to contact and adhesion of opposed villous surfaces. (Plastic exudation not material of adhesions. It may remain entangled and undergo caseous change—"tubercle of pleura," or become calcified, so-called "osseous plates.")</p> <p>General adhesions may obliterate pleural cavity. Local adhesions may incapsulate serum. Excess of serum—lung collapsed at hilus of lung. Too long collapsed, may be bound down by adhesions, expansion prevented, and air sacs agglutinated. Lesser adhesions—lesser results. Pleuritic serum may become purulent (empyema). Pleurisy often located near superficial tubercle. Tubercle perforating pleura causes pneumo-hydro-thorax.</p>
Causes.	<p>Acute—due to cold. Secondary—in blood states and acute disease. Local—co-existent with superficial tubercle</p>
Symptoms.	<p>1st Stage.—Chill, stitch in side, febrile action, incomplete restrained breathing. Irritable, dry cough. Later—pain relieved and dyspnoea developed, due to pressure of serum on lung and diaphragm. If—empyema frequent chills, weakness, foul tongue, sallow skin. Tubercle develops or typhoid state liable. If perforation—sudden, excessive dyspnoea; symptoms of collapse.</p>
Physical Signs.	<p>Depend on plasticity and movement of exudation. Rough surface gives a to and fro friction sound. Adhesions give every variety of fine and coarse rales, unaffected by cough. Layers of thickened pleura and exudation give percussion dullness and render voice and breath sounds muffled, vague and distant. Over serum, chest wall is full and immovable. Intercostal spaces bulge, percussion flat—high in pitch, short duration, wooden quality. Upper line of dullness abrupt, horizontal, changing with position of the body. Over serum, absence of voice and breath sounds. Aegophony at surface of fluid. Vocal fremitus wholly absent. Over compressed lung, sounds harsh, raised in pitch. In perforation, signs of serum below. Above, air gives tympanitic resonance on percussion sometimes pectoriloquy, and splashing of serum or succussion.</p>
Diagnosis.	<p>Friction double. Rales unaffected by cough. Dullness, with absence of voice and breath sounds and fremitus. Distinguished from pneumonia : Sounds conveyed in pneumonia. Crepitant rale in pneumonia. Distinguished from hydro-thorax : Both sides involved. Non-inflammatory.</p>
Prognosis.	<p>Many evanescent cases unsuspected. Most serous and sero-plastic cases recover. Excessive plasticity cripples lung. Permanent adhesions may cause local congestion or inflammation of lung, local or general emphysema. May, by causing irritation favor infiltration and caseous masses ("crude" yellow tubercle) in lung. Empyema often grave. Pneumo-hydro-thorax, bad.</p>
Treatment.	<p>Abortive.—Cups (dry) to side. Counter irritation active—prompt, cathartic (saline). Antiphlogistic—antispasmodic—anodynes for cough. When exudation is complete.—Aid process of absorption. No depressing measures. Food, quinine, iron, alcohol, fresh air, exercise, Absorption aided by mild counter-irritation. Ol. terebinthinae, or ungt. Iodini comp. : also by Iodide of Potassium. In slow absorption, perform paracentesis or aspirate.</p>

SYNOPSIS OF PNEUMONIA.

ENGLISH CLASSIFICATION.	GERMAN PATHOLOGICAL CLASSIFICATION
Pneumonia. Pneumonitis. Lobar Pneumonia. Inflammation of Lungs. Lung fever. (Popular names.)	Croupous Pneumonia.
Chronic Pneumonia..... } Cirrhosis of Lung..... }	
Capillary Bronchitis—with Lobular Pneumonia, and collapse of Lob- ules.....	Catarrhal Pneumonia.

PNEUMONIA.

PNEUMONITIS, INFLAMMATION OF THE LUNG, CROUPOUS PNEUMONIA.

Definition.	A local inflammatory disease of the lungs, usually idiopathic, with exudation of coaguable lump into the air sacs.
Pathology.	1st Stage.—Congestion, 2d Stage.—Red hepatization; solid, coagulated cell contents; white blood corpuscles (exudation corpuscles)—fibrin albumen, salts, haematin. Stasis in capillaries. 3d Stage.—Gray hepatization. Fatty metamorphosis of cell contents. Exudation removed by absorption and expectoration. Rarely leaves chronic consolidations. Often pigmentation due to transuded haematin-melanine. Usually (slight or great) co-existing local pleuritis.
Causes.	Age.—More often in adults. Sex.—More often in males. Exposure to damp and cold. Exhaustion. Secondary in acute fever, and septic blood condition.
Symptoms.	Marked chill or rigor—local stitch—rapid respiration, 30, 40, 60. Increased temperature—pulse full, frequent; may be active delirium. Bloody, "rust-colored," sputa. Short suppressed cough; anxious, painful face; dilatation of nares; circumscribed flush of cheek; asthenic cases and old age, may be no chill, little fever and no pain; delirium low, wandering; sputa dark, "prune juice," mahogany-colored; pulse weak and rapid, or slow. Assumes a typhoid form.
Physical Signs.	Differ in the three stages. Three stages usually co-exist in different lobes, hence physical signs vary in different parts of lung. 1st Stage. (Hyperæmia.) Slight dullness on percussion; tube sounds harsh; vesicular element of sound muffled. Early signs doubtful; with exudation crepitant rale present—fine crackles in inspiration only. 2d Stage. (Solidification.) Crepitus ceases; flatness on percussion (high pitch, short duration, wooden quality). Auscultation—bronchial breath and bronchial voice. Fremitus increased in partial pneumonia. Fremitus diminished in general or double pneumonia. 3d Stage. (Liquefaction—resolution.) Returned crepitant rale—"rale redux." Sub-mucous and mucous rales. Dullness on percussion. Gradual return of normal, "breezy," "vesicular," respiration. Crepitus of local pleurisy may co-exist.
Diagnosis.	Chill, fever, pain; circumscribed blush, rapid breathing, bloody sputa; flatness; crepitant rale; voice and breath bronchial—"rale redux."
Prognosis.	In healthy adults, recovery; resolution often by fifth day; the rule is in nine to twelve days. Weak and aged—typhoid form—often fatal. Very rarely develops phthisis.
Treatment.	May abort early by derivatives—diaphoresis, cathartics, cupping. Early period. Allay fever. Aconite, ammonia, fomentations, poultices, mild diet. Later period.—Prevent asthenia, favor resolution. Diet: Tonics, stimulants.

SYNOPSIS OF EMPHYSEMA.

PULMONARY, VESICULAR EMPHYSEMA.

Definition.	A local chronic disease, due to rarefaction of pulmonary structure and dilatation of air sacs.
Pathology.	May be general vesicular emphysema or often of upper lobes only, or confined to a few lobules only. Lungs do not collapse on opening thorax. May be distinct cysts, or poutings on the surface; feel soft, elastic. Progressive lesions, enlarged air sacs, obliterated alveoli, perforation of cell walls, cells coalesce, cysts of small or large size. Fatty degenerations and destruction of intercellular tissue. Capillaries obliterated; their blood leaves pigmentation. Obstructed lung circulation causes dilatation of right heart, hence general venous retardation, favoring chronic diseases of the viscera.
Causes.	Lobular emphysema, is "vicarious," caused in inspiration, compensating adjacent, atrophied, solidified, or collapsed lobules. General vesicular emphysema, always due to excess of inspired air, over air expelled in expiration as in chronic bronchitis, with thickening and relaxation of tubes. Usually fatty degeneration and atrophy of connective tissue. Emphysema of upper lobes only, due to expiratory force and obstruction at glottis; in pertussis, chronic cough, heavy lifting, gymnasts, porters, musicians, etc. Rarely fatty degeneration is primary. Very rarely due to rigid thorax.
Symptoms.	Dyspnœa, labored breathing, respiration and circulation easily disturbed. Often bad circulation, disease of heart, chronic disease of distant parts.
Physical Signs.	Lobules, not often detected. General, and upper lobes,—resonance vesiculo-tympanic. Respiratory sounds feeble. Expiration prolonged, blowing, low in pitch; variety of sibilant and sonorous rhonchi. Chest full, rotund; intercostal spaces full. Expansion slight. Rising of chest "en masse." Muscles of neck and chest fixed. Broad, short neck. General emphysema, diaphragm depressed, heart also resting on it, falls, becomes horizontal.
Diagnosis.	Heart apex outward; liver may be depressed. Diag. from asthma, chronic bronchitis (often co-exists).
Prognosis.	Can be alleviated; rarely cured.
Treatment.	Progress checked by removing cause; avoid cold. Cure laryngeal obstruction, also chronic bronchitis. Favor tone of elastic tissue by hygienics, diet, quinine, pot. iod.

SYNOPSIS OF PULMONARY CONSUMPTION OR PHTHISIS.

Pulmonary Consumption considered according to older pathology of school of Laennec, Louis, etc.

Definition.	Wasting disease of the lungs. A local expression of a blood state, of a specific diathesis. Due to tubercular deposits and their complications.
Pathology.	<p>1st Stage.—Tuberculization.—Deposit of tubercle usually yellow or crude; rarely miliary. Yellow tubercles coalesce, form caseous masses. Involved tissues atrophy. Caseous masses shrink; compensative emphysema; usually at apices co-existing localized pleurisy, bronchitis, pneumonia.</p> <p>2d Stage.—Softening.—Caseous masses become pultaceous by imbibition of serum, and pus due to ulceration and infiltration of adjacent tissue. Tissue destroyed, vessels obliterated or eroded; usually opening into bronchi.</p> <p>3d Stage.—Cavities.—Due to evacuation of tubercular pus. May be large or small, smooth or ragged. Wall may be exposed, lung tissue infiltrated with tubercles, exudation matter, or may be cicatricial tissue. Large vessels and bands of fibrous tissue may bridge over large cavities. Cavity may open freely into bronchus, or by a valvular opening. Cavities may contain mucus. Cavities may contract, granulate and heal.</p>
Causes.	<p>Great predisposing cause tubercular diathesis, usually hereditary; sometimes induced by environnement. Obscure relation to scrofulous, strumous and eczematous diathesis.</p> <p>Developing and exciting causes.—All depressing influences—indigestion, bad diet, privation of food, over-work, exposure to cold and damp, dissipation, close confinement at work in foul air, sedentary occupations, stooping, inhaling dust, indoor life, want of exercise and chest expansion, damp changing climate, low altitude, depressing diseases, as typhoid fever, measles, small-pox, syphilis.</p>
Symptoms of First Stage.	<p>1st Stage.—Variable; may be indigestion, debility, increased temperature, emaciation (loss of fat) and loss of strength. Short accelerated breathing, pallor, increase of temperature in the afternoon. May be early hæmoptysis (bronchorrhagia), early pleural plastic exudation, fugitive or fixed pains due to dry pleurisy. Cough and expectoration of local bronchitis; may be hoarseness.</p> <p>Inspection.—Small chest, sinking under clavicles, sinking in intercostal spaces. Frequent, shallow and feeble breathing.</p>
Physical Signs of First Stage.	<p>Mensuration.—Reduced circumference and expansion.</p> <p>Palpation.—Increased vocal fremitus, frequent breathing and feeble expansion.</p> <p>Percussion.—Dullness—slight or marked—raised in pitch, of short duration, of wooden quality. May be sense of resistance.</p>

SYNOPSIS OF PULMONARY CONSUMPTION, ETC.—*Continued.*

Physical Signs. (Continued.)	Auscultation.—Weakened respiration, absence of vesicular murmur, "bronchial breathing." Inspiration raised in pitch. Expiration prolonged and raised in pitch; bronchial voice, bronchial whisper and bronchial cough; increased vocal fremitus; respiration may be wavy, jerking, divided—cog-wheel respiration; heart sounds conveyed.
Second Stage.	2d Stage.—Hectic more marked; expectoration more free; contains muco-pus—pus, "tubercular pus," fibrous tissue, casts, shreds, etc.—(Pulmonary elements), fat granules, fatty cells, cholesterine. Sputa is nummular, <i>i.e.</i> , coin shaped masses at bottom of frothy fluid; decline of health, strength, flesh; loss of menses in this or early stage, or later; liability to hemorrhage from the eroded vessels; night sweats. Immediate Percussion.—"Myoidema." Auscultation.—Sub-crepitant rale over softened part.
Third Stage.	3d Stage.—Increased debility, emaciation, flatness of chest, short, quick, labored breath, bad appetite, weak stomach—nausea especially from fatty food—vomiting, reflex after cough. Cough hard and sputa copious, especially in the morning.
Symptoms.	Debility increased by severe afternoon hectic, profuse night sweats, excessive expectoration, anorexia. Sometimes colliquative diarrhœa; face sunken; cadaveric, eyes staring; breath gasping; tongue dry and brown; fingers clubbed; liability to severe or fatal hemorrhage, due to erosion of vessels in cavities.
Physical Signs.	Inspection—May be flatness—retraction. Percussion—May be dullness; or resonance over large superficial cavities; cracked pot sounds. Auscultation—Cavernous, amphoric respiration, voice whisper and cough; gurgles, pectoriloquy, aegophony, metallic tinkle.
Diagnosis.	From chronic bronchitis and emphysema.
Prognosis.	Always uncertain and guarded. Early stages most favorable, yet extreme cases may be checked. Prognosis determined by temperament, family history, habits, treatment and climate.
Treatment.	To prevent, cure, arrest, and to prolong life: Preventive.—Attend to ventilation, digestion, clothing, exercise, occupation, chest expansion, out-door life, climate. Lessen cough and secretion.—Anti-spasmodics, balsamics, anodynes, counter irritants. Regulate digestion and bowels.—Create appetite, aid digestion, support strength. Bismuth, bark, pepsine, milk, cream, butter, kumyss, peptonized milk, fats, ol. morrhuae, glycerine, pancreatic emulsion, wine, whiskey, etc., hypophosphites, etc. Stop fever and night sweats.—Quinine, sulphuric acid, ergot, atropine; sage infusion, "dry salted towels." Relieve fugitive pain.—Anodynes, counter irritants. Relieve occasional vomiting.—Check diarrhœa.

HEART DISEASES.

HEART.

Location.—Upper border of third left costal cartilage to lower border of sixth, one-half to three-quarters inch to right of sternum, extends to left to half inch within the nipple.

Deep Cardiac Region.	{	Occupies all of lower sternal region.
		“ “ part of right mammary region
		“ “ left “
Superficial Cardiac Region.	{	“ slightly right and left infraclavicular region.
		Part not over-lapped by lung—a triangle whose apex is at fourth left sternal articulation, extends downwards and outwards to the union of fifth rib with cartilage, and inward and downwards to sixth rib.

LOCATION OF HEART VALVES.

Pulmonary.—Left edge of sternum near lower edge of third cartilage.

Aortic.—A little lower, midway from edge to median line.

Tricuspid.—Middle of sternum, oblique from third left to fourth right interspace.

Mitral.—One quarter inch below aortic. Horizontally across sternum opposite fourth left articulation.

ANATOMY OF HEART.

Distance of mitral from tricuspid, one-half inch.

A radius of one-half inch includes a part of all valves.

Behind.—Apex corresponds to seventh to eighth rib in left vertebral groove.

WEIGHT.

Average male, $9\frac{1}{2}$ oz. (Walshe.)

“ 10–12 oz. (Gray.)

Female, 8–10 oz. “

SIZE.

Length.....5 inches.

Width..... $3\frac{1}{2}$ “

Thickness..... $2\frac{1}{2}$ “

Thickness of right auricle . . .	1 line ($\frac{1}{12}$ of an inch.)
“ “ left “	$1\frac{1}{2}$ line.
“ “ septum	4 lines.
“ “ right ventricle . . .	$2\frac{1}{2}$ to 3 lines.
“ “ left “	4 to 5 “
Capacity of right ventricle . . .	2 fl. oz.
“ “ left “	2 fl. oz.

METHODS OF EXAMINATION OF THE HEART.

1. *Inspection*.—Note the location and character of impulse or “apex beat.”
2. *Mensuration*.—Note relative distance of nipple from median line on two sides.
Note vertical distance, on two sides, from top of third rib to lower edge of sixth.
3. *Palpitation*.—1. Note præcordial motion.

(a.) Force	}	Of Impulse.
(b.) Duration		
(c.) Rapidity		
(d.) Character		

 2. Synchronism of impulse and systole of heart.
4. *Percussion*.—Map out area of deep cardiac dullness.
Superficial area of cardiac dullness.

By mediate	}	Percussion.
By immediate		
By auscultatory		
5. *Ascultation*.—Observe frequency of action.
Rhythm of action.
Duration of first sound.

“ “ “	rest.
“ “	second sound.
“ “ “	rest.

RHYTHM, DURATION AND CHARACTER OF NORMAL CARDIAC SOUNDS.

Whole period of heart's action, 10 :

1st sound	4
1st rest	1
2d sound	2
2d rest	3—10

First sound synchronous with systole and apex impulse.

Second " " " diastole (beginning).

At apex, first sound—ou bb.—second sound, dup.

At base, " " —up. — " " tup.

(Walshe.)

Apex and over ventricles—llug. —second sound, te.

(Leaming.)

THEORIES OF CAUSE OF CARDIAC SOUNDS.

(1.) First sound, due to

1. Muscular sussurrus and friction in cardiac wall.
2. Blood motion (inter se)
3. " " against ventricular wall, columnae and chordae.
4. Vibration of valves (mitral).
5. " " chordae tendinae.
6. " " chest wall.
7. Impulse of heart.
8. Præcordial friction.

The first sound is probably due to each and all of the above causes—factors of a composite first sound.

2. The second sound is also composite, due to the synchronous closure of the aortic and pulmonary valves.

The first sound is best heard over body of heart and over apex, that is in the superficial cardiac region.

The second sound is best heard at the base over the valves.

FUNCTIONAL DISEASES OF THE HEART.

1. Anæmic blood murmurs.

2. Neuroses.	{	Hyperæsthetic.	{	Intermission.
				Irregularity.
				Palpitation.
				Spasm.
	{	Atonic.	{	Cardiac Tremor.
				Syncope.
				Paralysis.
				Angina Pectoris.

(1.) ANÆMIC BLOOD MURMURS.

In anæmia, the blood is in an impoverished, watery condition. Women who are pale, destitute of color in the face, who suffer with cold feet, uterine disorders, globus hystericus, are the most frequent subjects.

Motion of the blood *inter se* produces in the heart and great vessels a soft blowing sound, which may also range in intensity from a gentle to a very violent murmur; this anæmic or hæmic murmur, however, usually lacks the friction, harsh grating, sawing character of organic murmurs. It is best heard at the base of the heart, at the junction of the third left cartilage, with the sternum, and is also heard throughout the large arterial trunks, the innominate and sub-clavian arteries—and down the sternum to the tip of the xyphoid. The sound is carried simply by convection. In cases of extreme anæmia, there are also venous murmurs in the neck—a continuous humming, called “bruit du diable.”

DIAGNOSIS OF ANÆMIC MURMURS.

1. Character—soft and blowing.
2. Originated at base and carried by large vessels.
3. Occur in anæmic persons.
4. Loudest when person exercises violently.

(2.) NEUROSIS OF THE HEART.

Disorders due to nerve causes.

Nerve power of heart.—The heart is an involuntary muscle, which receives a constant supply of motor power from the pneumo-gastric nerve, and the superficial and deep cardiac ganglia.

All over the surface of the heart, and in its substance, are numerous little ganglia, connected together and constantly supplying stimulus for the heart's action and rhythm.

IRREGULARITY AND INTERMISSION.

The most common of all causes of irregularity, intermission, and palpitation, is indigestion. Irritation of one branch of pneumo-gastric nerve—the gastric,—disorders the other branches, and produces palpitation and dyspnoea.

Excessive use of tea and coffee, and tobacco especially, is a cause,—the latter acts not only by producing dyspepsia through excessive action of the salivary glands, but directly through the nervous system.

Alcoholic habit causes first a violent, then an irregular heart action. Fright, excitement, excessive mental effort, great fatigue from overwork, or absence of sleep, will give rise to irregularity in the heart's action.

SPASM.

The action of the heart in a child who has St. Vitus's dance is typical cardiac spasm. This is the condition in which is often heard an intra-ventricular or functional apex murmur. This is produced by irregular contractions of the columnae carnae and layers of cardiac muscle, and is heard either as a modification of the first sound, or at the completion of the second sound, at the apex.

TREMOR.

When a person has indulged in dissipation until the nervous system is exhausted, there will be a tremulous action of the heart, discernible by palpation.

SYNCOPE.

Syncope is that form of fainting which is due to failure of the heart's action. The cessation may be temporary, with temporary cerebral anæmia, from failure of the heart to send blood to the brain, or the cardiac relaxation may be complete and death ensue. The failure to contract is usually due to deficient nerve force. If merely transient, its cause being a temporary derangement of the nerve centres, we have vertigo, unconsciousness, feeble action of heart, or action altogether inappreciable, face suffused; after a brief time the heart resuming its work. If syncope continue a long time, a heart clot may form; this will give rise to a murmur for a considerable length of time; it may lead to embolism, the embolism occurring months later.

PARALYSIS OF THE HEART.

Shock or injury to the sympathetic nerve may be so great that it never reacts to supply stimulus for the heart's action.

Diphtheria is a cause of paralysis of the heart, also cerebro-spinal meningitis and typhus fever.

ANGINA PECTORIS

Is placed among neuroses, because its lesion is not known. (All its symptoms may occur in organic disease.) Its pathology is supposed to be in many cases embolism or occlusion of the coronary vessels.

ORGANIC DISEASES OF THE HEART.

List of organic diseases of the heart :

Anæmia—congestion.

Endocarditis—having valvular lesions. ^(1.)

Pericarditis.

Myocarditis.

Hydro-pericardium.

Atrophy.

Hypertrophy.

Dilatation—having valvular lesions. ^(2.)

Fatty degeneration.

Atheroma—having valvular lesions.^(3.)

Hemorrhage.

Embolism.

Aneurism.

Rupture.

Injuries.

Malformations—having valvular lesions.^(4.)

Syphilis.

Tubercle.

Cancer.

Anæmia and congestion are hardly to be regarded as distinct diseases, being disturbances of cardiac vascularization.

VALVULAR DISEASES OF THE HEART.

Systolic.	<p>(1.) SYSTOLIC.</p> <p>(a.) Left side of heart :</p> <ol style="list-style-type: none"> 1. <i>Aortic obstruction</i>.—Obstacle to outward flow of blood through aorta orifice. 2. <i>Mitral regurgitation</i>.—Leakage of blood through mitral valve into left auricle. <p>(b.) Right side of heart :</p> <ol style="list-style-type: none"> 1. <i>Pulmonary obstruction</i>.—Obstacle to outward flow of blood through pulmonary orifice. 2. <i>Tricuspid regurgitation</i>.—Leakage of blood through tricuspid valve into right auricle.
Diastolic.	<p>(2.) DIASTOLIC.</p> <p>(a.) Left side of heart :</p> <ol style="list-style-type: none"> 1. <i>Aortic regurgitation</i>.—Leakage of blood through aortic orifice into left ventricle. 2. <i>Mitral obstruction</i>.—Obstacle to flow of blood from left auricle to left ventricle. <p>(b.) Right side of heart :</p> <ol style="list-style-type: none"> 1. <i>Pulmonary regurgitation</i>.—Leakage of blood through pulmonary orifice into right ventricle. 2. <i>Tricuspid obstruction</i>.—Obstacle to flow of blood from right auricle to right ventricle.

CARDIAC HYPERTROPHY.

Definition.	Enlargement of the heart as a whole, or of the walls of any one of its cavities—the result of increased effort of heart.
Pathology.	<p>Hypertrophy of left ventricle most often—more often than right ventricle; rarely auricles. Increase of weight, size, thickness of walls.</p> <p>Concentric hypertrophy: Thickening of walls at expense of cavity.</p> <p>Excentric hypertrophy: Hypertrophy with dilatation.</p> <p>Due to hypertrophy of existing muscular fibres.</p> <p>Possibly new fibres developed.</p> <p>Hypertrophy, at first a conservative process, changes to dilatation and fatty metamorphosis after long standing.</p>
Causes.	<p>Of left ventricle :</p> <p>Aortic valvular lesions.</p> <p>Mitral valvular lesions.</p> <p>Atheroma of aorta and arteries.</p> <p>Obstructed circulation in viscera.</p> <p>Pericardial adhesions.</p> <p>Habitual violent work or exercise.</p> <p>Of right ventricle :</p> <p>Emphysema and other chronic lung disease; obstructive pulmonary valvular disease.</p> <p>Mitral lesions. (Indirectly.)</p>
Symptoms.	<p>Full, strong pulse in hypertrophy of left ventricle. Liability to congestion of brain if the heart is over active.</p> <p>With hypertrophy of right ventricle, a slow venous circulation, dyspnœa, co-existing lung disease.</p>
Physical Signs.	<p>Inspection.—Fullness of precordial region, and heaving apex beat. Apex lower and outward—hypertrophy of left ventricle.</p> <p>Mensuration.—Nipple carried outward; increased distance from third to sixth rib.</p> <p>Palpation.—Detect violent apex beat—and changed location.</p> <p>Percussion.—Increased area of dullness to the left, and down, for the left ventricle, to right of sternum for right ventricle.</p> <p>Auscultation.—Detect valvular murmurs of lesion, causing hypertrophy. Hear accentuated first sound. Hear sharp, loud, abrupt click of second sound.</p>
Diagnosis.	Precordial fullness; increased area of dullness; full, strong pulse; strong impulse; displaced apex; valvular lesion; intensified normal second sound, due to aortic resiliency.
Prognosis.	Prognosis in simple hypertrophy good, if life be regular and no increase of lesion. Bad, if hypertrophy is associated with dilatation. Danger of cerebral congestion, dilatation, fatty degeneration, etc.
Treatment.	<p>Quiet life; light work; temperance; plain diet.</p> <p>Keep excretion free.</p> <p>Avoid causes of increased lesion.</p>

CARDIAC DILATATION.

Definition.	Enlargement of the heart, or one or more of its cavities, with thinning of their walls.
Pathology.	Increase of size of heart ; cavities increased in size and capacity ; walls thin, often in state of fatty degeneration ; lesion of mitral or aortic valve as a rule ; valve may be separated secondary to dilatation ; rarely hypertrophy co-exists, second in order, and conservative ; more often walls are thin, even as parchment (rare).
Causes.	Mechanical internal dilating power of blood during diastole (rest—relaxation of heart). Usually due to mitral regurgitation or aortic regurgitation ; also due to softening, relaxation, fatty degeneration of heart walls, in acute fevers of low malignant type.
Symptoms.	Weak pulse—often intermittant, irregular ; slow, imperfect venous return circulation ; dyspnœa ; bad digestion ; passive congestion of lungs, kidneys and liver ; cold extremities ; pale or cyanosed face ; liability to syncope ; oedema of lungs ; general anasarca.
Physical Signs.	Precordial fullness ; weak, heaving, wavy motion over heart ; not a strong defined impulse ; nipple carried outward ; increased distance from third to sixth rib ; increased area of dullness ; heart sounds weakened ; loud abnormal sounds indicating mitral regurgitation or aortic regurgitation.
Diagnosis.	Weak pulse ; weak, wavy impulse ; large area of dullness ; regurgitation, dyspnœa ; attacks of syncope ; intermission and irregularity of pulse ; general weakness ; secondary disorders of other organs.
Prognosis.	Always bad.
Treatment.	A quiet life ; avoid heavy work, active exercise, excitement, dissipations, and indigestion. Preserve full action of bowels, kidneys and skin. Strengthen health and action of heart by plain food, quinine, strychnine, and iron. Regulate heart's action by digitalis, convallaria, cactus. Combat cardiac failure by alcohol, morphine, Hoffman's anodyne, nitrite of amyl.

FATTY HEART.

QUAIN'S DEGENERATION.

Definition.	A destructive metamorphosis of the muscular structure of the heart. (Distinguished from mere deposition of adipose on the heart of obese persons.)
Pathology.	Heart.—Pale yellow, soft, small (unless dilatation co-exist). Striae of muscular fibres grow faint, disappear; replaced by fatty granules, oil globules and atheroma.
Causes.	Indolent life; alcoholism; old age; acute fever of low type; climacteric changes, atheroma of coronary arteries; embolism of coronary arteries.
Symptoms.	Weak circulation; feeble pulse; cold extremities; pallor, cyanosis; weak heart impulse; liability to syncope; cerebral anæmia. Weak heart sounds, intermission, irregularity; dyspnœa, sighing, irregular breathing. (Cheyne's symptom.) (Co-existing arcus senilis, atheroma of arteries in old people.)
Physical Signs.	No precordial prominence; no perceptible apex beat. May be reduced area of dullness; sounds feeble, often distant.
Diagnosis.	Age, general health, habits, arcus senilis, weak pulse, often syncope, faint heart sounds.
Prognosis.	Bad. Liability to death by syncope.
Treatment.	Arrest progress by good diet, temperance, tonics. Avoid syncope; avoid dilatation; avoid sudden or great effort. Treat syncope by stimulants.—Alcohol, ammonia, Hoffman's anodyne, amyl, etc.

ENDOCARDITIS.

Definition.	Inflammation of the endocardium.—A local disease usually secondary to and complicating overloading of blood with irritant morbid material.
Pathology.	Removal of epithelium.—Surface hazy, granular; congestion of sub-serous vessels; development of villi; infiltration of tissue producing roughened, thickened, villous surface, or masses (vegetations). May be hair-like processes, or pendunculated polypoid bodies. Inflammatory products may be re-absorbed or remain; may be fibrous or become calcareous; may cause atrophy, hypertrophy, distortion, retraction, etc., of valves. Polypoid masses may be detached and cause embolism of other viscera. Endocarditis usually on left side of heart only. Endocarditis of foetus in utero, on right side of heart. Vegetations may grow from fibrin from the blood.
Causes.	Rheumatism, Bright's disease, scarlatina. Rarely typhus, typhoid, variola, rubeola, diphtheria, sepsis.
Symptoms.	Often none. May be dyspnœa, irritable action of the heart. Often not diagnosed.
Physical Signs.	Sounds induced by valvular lesions. Aortic direct friction; mitral murmurs. Murmurs disappear if absorption results; murmurs increase with extent of lesion.
Diagnosis.	By daily auscultation, during course of causative diseases, to detect friction murmurs.
Prognosis.	Not immediate danger. Danger of valvular disease.
Treatment.	Treatment of primary disease. Subsequent use of quinine, iodide of potassium, and mercury to promote absorption.

PERICARDITIS.

Definition.	Inflammation of the serous pericardium.—A local inflammation rarely idiopathic or traumatic ; usually the result of overloading the blood with irritant, morbid matter.
Pathology.	Congestion of sub-serous vessels; removal of epithelium ; surface hazy, granular ; reddened patches; development of granulations; vascular villi; friction of surfaces; liable to adhere; patches of complete union; thin bands, fibres, etc., due to contact of granulating surfaces. Absorption may be complete; may leave white patches on heart, hairy processes, fine or coarse adhesions. Serum compresses heart, distends pericardium. Serum absorbed, surfaces again in contact. Myocarditis may co-exist.
Causes.	Rheumatism, Bright's disease, scarlatina; other acute fevers; rarely traumatic—rarely idiopathic (catching cold).
Physical Signs.	Early.—To and fro pericardial friction murmurs ; heart sounds increased, apex beat violent, pulse full—fast. With effusion.—Fullness of precordial region ; increased area of dullness; friction sound lost; heart sounds feeble; may be distant. Later.—Muffled friction may return ; normal action of heart and normal area of dullness.
Diagnosis.	To and fro friction ; area of dullness. [Hydro-pericardium has no friction sound, no fever, and co-exists with general dropsy.]
Prognosis.	Generally good. Pericardial adhesions may cause hypertrophy.
Treatment.	Treat primary disease. Locally—anodyne, alkaline fomentations for pain. Lower frequency of pulse.—Aconite, digitalis, veratrum. Later.—Quinine, iodide of potassium, and mercurials for absorption.

CARDIAC MURMURS.

	FUNCTIONAL.
(1) Functional.	<p>1. <i>Systole of heart</i>— Anæmic murmurs; blowing in quality; heard at base of heart and diffused over chest, especially in course of vessels. Intraventricular murmur, heard at early part of systole at apex.</p> <p>2. <i>Diastole of heart</i>— Re-duplicated second sound heard at aortic and pulmonary orifice; due to nervous action of two sides of the heart; resiliency of aorta and pulmonary arteries unequal, aortic and pulmonary valves do not close synchronously.</p>
	ORGANIC.
(2) Organic.	<p>1. <i>Systole of heart—Left ventricle</i>— 1. <i>Obstructive</i>.—Obstruction at aortic valves, friction, etc.; heard at aortic orifice and over arch of aorta (at third left and second right sterno-costal articulation.) 2. <i>Regurgitation</i>.—Insufficiency of mitral valve; regurgitation of blood into left auricle; heard at apex in front. May also be heard around left side and behind in the left vertebral groove, from the fifth to eighth ribs.</p> <p><i>Right ventricle</i>— 1. <i>Obstructive</i>.—Obstruction at pulmonary valves; friction murmur, superficial, intense, localized over valve—third left sterno-costal articulation, may exist behind second left cartilage. 2. <i>Regurgitant</i>.—Insufficiency of the tricuspid valve; heard at mid-sternum, opposite third or fourth cartilages.</p> <p>2. <i>Diastole of heart—Left ventricle</i>— 1. <i>Obstructive</i>.—Obstruction at mitral valve; friction murmur heard at apex only. Præ systolic, <i>i.e.</i>, in latter part of diastole. 2. <i>Regurgitant</i>.—Insufficiency of aortic valves; murmur heard at base, and conveyed down sternum to apex of heart replaces second sound.</p> <p><i>Right ventricle</i>— 1. <i>Obstructive</i>.—Obstruction at tricuspid valve; friction of blood coming from auricle to ventricle; murmur heard over central cardiac area. 2. <i>Regurgitant</i>.—Insufficiency of pulmonary valves; heard over pulmonary valves and down right side of heart.</p>
	ANÆMIC MURMURS.
Where occurs.	<p>Occur in an anæmic woman mainly. " " feeble and aged. " " pregnancy. " " course of blood disease. " " convalescence.</p>
When heard.	<p>Heard during systole only. " widely diffused over chest. " loudest at base of heart. " over arch of aorta and (transmitted by convection) over thoracic aorta down side of sternum to zyphoid cartilage; over innominate, carotids, sub-clavian. " loudest when patient is fatigued, active or excited. " less when quiet. Diminished by tonic treatment, digitalis, test.</p>
Character.	<p>Character.—Soft, blowing, or loud violent systolic murmur at base. May have grating, filing, rasping, sawing musical qualities. Distinguished from organic by absence of friction, and by test of time and treatment. Accompanied with symptoms of anæmia, debility, indigestion, hysteria, etc.</p>

CARDIAC MURMURS.

TABLE OF SYNONYMOUS TERMS.

1. Aortic. Systolic. Obstructive. Direct.	3. Aortic. Diastolic. Regurgitant. Insufficient. Indirect.
2. Mitral. Systolic. Insufficient. Regurgitant. Indirect.	4. Mitral. Diastolic. Obstructive. Direct. Præsystolic.

The most frequent and important organic murmurs to recognize are:

1. Aortic systolic (obstructive, direct).
2. Mitral systolic (regurgitant, indirect).
3. Mitral præ systolic (obstructive, direct). Very much less frequently we meet with
4. Aortic diastolic (regurgitant, indirect).

The above are all murmurs of the left side of the heart, products of endocarditis, atheroma, traumatism, and resulting from rheumatism, uraemia and acute diseases.

Of the murmurs of the right side of the heart, two are definitely recognizable, but very rare, viz.:

5. Pulmonary systolic (obstructive, direct.)
6. Tricuspid systolic (regurgitant, indirect).
7. Tricuspid præ systolic (obstructive, direct) is pathologically and theoretically possible, but not likely to be diagnosed.
8. The pulmonary diastolic (regurgitant, indirect) is likely to be detected only when co-existent with well defined pulmonary systolic murmur.

ANEURISM OF THORACIC AORTA.

1. Of ascending arch within pericardium.
2. Of junction of ascending and transverse arch.
3. Of transverse arch.
4. Of descending arch.
5. Descending aorta within thorax.

DEFINITION.—Aneurism—aneuruno, to dilate—a local dilatation of the aorta, forming a tumor containing blood, and having walls, the remnant of the vessels walls, and its investing fibrous sheath usually hypertrophied.

PATHOLOGY AND AETIOLOGY.—Usually alteration by endarteritis, atheroma and calcification of wall of artery, and atrophic weakening; syphilis, rheumatism, gout and local injury lead to these degenerations. Aneurism may be fusiform, sacculated or dissecting. The cavity may be full of fluid blood, or be partially or wholly filled with laminated fibrin.

The aneurism, by pressure, may set up inflammation of adjacent part, as of apices of lungs, pleura, bronchi. It may cause absorption of ribs and costal cartilages anteriorly, or of vertebrae posteriorly; may compress trachea, bronchi, oesophagus, vena cava ascendens, innominate vein. The sac may rupture into the pleura, trachea, bronchi, pericardium—or externally, beneath or through eroded integument.

SYMPTOMS.—Aneurism of ascending portion as a rule produces irritation of apex of right lung and its investing pleura, evidenced by cough, expectoration, local discomfort, dyspnoea, local lancinating and pulsatory pain.

Aneurism of ascending and transverse, also involves right recurrent laryngeal, inducing laryngeal spasm, irritability and aphonia.

Aneurism of ascending portion may obstruct right pulmonary bronchi or bronchus to upper lobe of right lung. Aneurism of transverse portion compresses trachea, cutting off air to lung and irritating both recurrent laryngeals.

Orthopnœa, laryngeal spasm, shallow chest expansion, suprasternal and epigastric sinking, anorexia often marked.

Aneurism of descending arch disturbs only left recurrent laryngeal and the apex of left lung, its investing pleura and bronchi—discomfort is then on left side of sternum and in the left scapular and interscapular regions.

PHYSICAL SIGNS.—INSPECTION.—In early stage aneurism may show nothing. Later, vibration and bulging are observed in second intercostal space to right of sternum, or, if transverse, pulsation may appear in suprasternal notch and extend up cervical vessels. Aneurism of descending portion, the bulging is to left of sternum, at second cartilage. Later, sternum, ribs and cartilages may bulge over aneurism or be eroded and the spheroidal pulsating tumor is seen to right or left of sternum.

PALPATION.—Vascular swelling and cyanosis of head, neck, arms and thorax from pressure on vena cava descendens—of right side only, if pressure on innominate vein. Vibratile pulsation, or “thrill,” is detected in intercostal spaces or through sternum, or in suprasternal notch; after erosion, tumor gives distensile sensation.

PERCUSSION.—Area of dullness in upper sternal region, extending to right or left or up into suprasternal notch.

AUSCULTATION.—ANEURISM OF ASCENDING ARCH.—Double bruit on right side of sternum, in second space, both sounds of friction, rasping, sawing, or whistling, ringing, booming, musical quality; sounds measureably conducted down the sternum and up into the cervical vessels. The second sound of heart usually discernible to left of sternum thus differentiating double aortic murmur. Pressure on apex may have developed local plastic pleuritic rales, local pneumonic crepitus, or stridulus by pressure on bronchus; respiratory sounds lessened or absent in lung.

ANEURISM OF TRANSVERSE ARCH.—Double bruit, heard with stethoscope, deep in suprasternal notch, over upper end

of sternum and up vessels of neck. Heart sounds heard separately. Tracheal stridulus very distinct at point of pressure, and the respiratory sounds very feebly developed throughout both sides of chest.

ANEURISM OF DESCENDING ARCH.—Double bruit heard of “aneurismal quality,” to left of sternum, above base of heart; but best heard in back, in left vertebral groove, and in interscapular region.

DIAGNOSIS.—By tumor, area of dullness, pulsating, distensible and thrilling sensations to touch, by characteristic double bruit. When present, by associated symptoms of pressure on air passages, pleura, lung apex, vertebrae and nerves; also by exclusion of cardiac origin of tumor, thrill and bruits.

PROGNOSIS.—Always unfavorable; aneurism may rupture externally or internally into lung, pleura, bronchi, trachea or pericardium. Death also by inanition, asphyxia, exhaustion.

TREATMENT.—Diminish arterial tension by quiet living, by open condition of bowels, by avoiding overloaded stomach, avoiding emotional excitement, fatigue and alcoholism. Enforced rest, cardiac sedatives and exclusive meat diet favor hyperinosis and filling of sac with laminated fibrin.





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